A high fat meal after peat smoke inhalation unmasks latent cardiopulmonary responses in rats

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Stress tests are used clinically to uncover underlying disease and predict future cardiovascular risk. Previously, we used treadmill exercise stress in rats to reveal latent effects of air pollution inhalation. Other daily stressors, when modeled experimentally, may have similar utility. For example, consumption of a high fat (HF) meal causes transient vascular dysfunction, increases in LDL cholesterol, and oxidative stress. Given the ubiquity of Western diets, the aim of this study was to assess the effects of air pollution exposure on cardiovascular responses to a HF challenge. Healthy male Wistar Kyoto rats were exposed once (1-hr) to filtered air (FA), or low (LP; 0.36 mg/m3 particulate matter) or high concentrations (HP; 3.30 mg/m3) of smoldering peat biomass smoke, a key wildland fire air pollution source. Rats were fasted overnight, then given an oral gavage of a HF suspension (60 kcal% from fat), mimicking a HF meal, 24-hr post-exposure. High frequency ultrasound to assess cardiac and superior mesenteric artery function, measurements of systemic lipids, hormones, and pulmonary and systemic inflammatory markers, and flow cytometry to assess circulating monocyte phenotype were carried out in un-gavaged rats, and/or in rats 2-hr or 6-hr post-gavage. Few effects were evident after peat exposure or gavage, alone. By contrast, exposure to LP increased heart isovolumic relaxation time and decreased serum glucose and insulin levels relative to FA 2-hr post-gavage and increased CD11 b/c-expressing monocytes 6-hr post-gavage. Exposure to HP increased serum total cholesterol, alpha-1 acid glycoprotein, and alpha-2 macroglobulin ($p = 0.063$) relative to FA 2-hr post-gavage and decreased serum corticosterone levels relative to FA ($p = 0.085$) at 6-hr post-gavage. Lastly, exposure to either LP or HP increased lung gamma-glutamyl transferase levels relative to FA 2-hr post-gavage. These data demonstrate the utility of HF challenge in revealing effects of air pollution that would otherwise be imperceptible, particularly at low exposure levels, and suggest exposure primes the body to heightened responses to pro-inflammatory triggers. This abstract does not reflect US EPA policy.